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=> s coffey-arthur.in.
L1            0 COFFEY-ARTHUR.IN.

=> s arthur.in
L2         2715 ARTHUR.IN

=> s coffey-arthur.in
L3            0 COFFEY-ARTHUR.IN

=> s coffey (2a) arthur.in
L4            0 COFFEY (2A) ARTHUR.IN

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L5            0 L2 AND WOUND (3A) (CARE OR DRESSING OR BANDAGE OR COVER)

=> s arthur and wound (3a) (care or dressing or bandage or cover)
L6            0 ARTHUR AND WOUND (3A) (CARE OR DRESSING OR BANDAGE OR COVER)
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=> d l10 12 ibib kwic

L10 ANSWER 12 OF 12 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 92200603 MEDLINE
DOCUMENT NUMBER: 92200603 PubMed ID: 1551208
TITLE: Neutralization of endogenous tumor necrosis factor ameliorates the severity of myosin-induced myocarditis.
AUTHOR: Smith S C; Allen P M
CORPORATE SOURCE: Department of Internal Medicine, Washington University School of Medicine, St. Louis, Mo 63110.
CONTRACT NUMBER: AI-31238 (NIAID)
SOURCE: CIRCULATION RESEARCH, (1992 Apr) 70 (4) 856-63.
Journal code: 0047103. ISSN: 0009-7330.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199204
ENTRY DATE: Entered STN: 19920509
Last Updated on STN: 19920509
Entered Medline: 19920424
AB . . . the inflammatory response. Using a murine model of autoimmune myocarditis, we studied the role of TNF and IFN-gamma in myocardial **inflammation**. **Neutralizing monoclonal antibodies** against **TNF**-alpha/beta and IFN-gamma were administered to myosin-immunized A/J mice to assess the effect on the severity of myocardial inflammation. Anti-TNF treatment. . .

=> d l10 1-11 ibib kwic

L10 ANSWER 1 OF 12 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001066282 MEDLINE
DOCUMENT NUMBER: 20557224 PubMed ID: 11105596
TITLE: [Treatment of Crohn disease in adults with tumor necrosis factor-alpha (TNF-alpha) antibodies].
Traitement de la maladie de Crohn de l'adulte par anticorps anti-tumor necrosis factor-alpha (TNF alpha).
AUTHOR: Belaiche J; Louis E
CORPORATE SOURCE: Service de Gastro-Enterologie, CHU de Liege.
SOURCE: REVUE MEDICALE DE LIEGE, (2000 Sep) 55 (9) 827-32. Ref: 28
Journal code: 0404317. ISSN: 0370-629X.
PUB. COUNTRY: Belgium
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001228
AB . . . synthesized by monocytes, macrophages, and T cells. TNF alpha plays an early central role in the cytokine cascade of the **inflammatory** process. Recently, chimeric **monoclonal antibodies** that **inhibits TNF** alpha have been used in the treatment of Crohn's disease. Infliximab has been the most largely used antibody. It is. . .

L10 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:652439 CAPLUS
DOCUMENT NUMBER: 134:157051
TITLE: Gene therapy targets for rheumatoid arthritis

AUTHOR(S): Gould, David J.; Chikanza, Ian C.; Chernajovsky, Yuti
CORPORATE SOURCE: Bone and Joint Research Unit, St. Bartholomew's and Royal London School of Medicine and Dentistry, Queen Mary and Westfield College, London, EC1M 6BQ, UK
SOURCE: Emerging Therapeutic Targets (2000), 4(4), 481-495
CODEN: ETTAF7; ISSN: 1460-0412
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 97 THERE ARE 97 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A review with 97 refs. is given on important developments in gene therapy for rheumatoid arthritis (RA). RA is the most common chronic systemic autoimmune inflammatory disease whose pathogenesis is not fully understood. The physiol. of inflammation was systematically studied and has provided specific targeted strategies for the modulation of inflammation. A no. of biol. agents targeted at reducing the inflammatory cascade of pathophysiol. reactions were developed. Some, such as interleukin-1 receptor antagonist (IL-1Ra), antitumor necrosis factor (TNF) .alpha. antibodies and TNF sol. receptors, were tested and are now in use clin. The clin. effects that were obsd. are transient, necessitating repeated treatments. Advances in mol. biol. have opened ways for the development of gene therapy in which specific genes are introduced, using either viral or non-viral ex vivo and in vivo gene transfer techniques, to locally enhance in vivo gene expression or suppress gene(s) of interest with a view to down-regulating inflammatory responses. The proof of concept was provided in a no. of animal models of inflammatory arthritis. Strategies for prodn. of cytokine **inhibitors**, such as **sol. TNF receptors**, or anti-**inflammatory** cytokines, such as IL-4, IL-10, transforming growth factor .beta. (TGF-.beta.), and interferon .beta. (IFN-.beta.), were developed. Other approached involve the regulation of cartilage and bone erosion using IL-1Ra and tissue inhibitors of metalloproteinases, modulating apoptotic pathways in the rheumatoid synovium and the use of decoy oligonucleotides to nuclear factor .kappa.B (NF-.kappa.B), whose local application was shown to be effective in down-regulating joint inflammation in rat models of arthritis. Cytokines and other mediators play important physiol. roles in the host's defense system against infections and malignancy. Their chronic inhibition or their constitutive expression by gene therapy may lead to the development of side effects. Thus, carefully regulated gene expression during long-term studies will be required to assess the safety of selective targeting of processes involved in inflammation.

L10 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
ACCESSION NUMBER: 1998:47304 CAPLUS
DOCUMENT NUMBER: 128:175964
TITLE: Ro 45-2081, a **TNF** receptor **fusion protein, prevents inflammatory** responses in the airways
AUTHOR(S): Gater, P. R.; Renzetti, L. M.
CORPORATE SOURCE: Hoffmann-La Roche Inc., Nutley, NJ, 07042, USA
SOURCE: Agents and Actions Supplements (1998), 49(Therapeutic Strategies for Modulating the Inflammatory Diseases), 67-71
CODEN: AASUDJ; ISSN: 0379-0363
PUBLISHER: Birkhaeuser Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Ro 45-2081, a **TNF** receptor **fusion protein, prevents inflammatory** responses in the airways

AB The **TNF** receptor **fusion protein**, Ro 45-2081, **inhibited** allergic and non-allergic **inflammatory**

responses in the airways. Treatment of sensitized guinea-pigs with Ro 45-2081 reduced allergen-induced influx of inflammatory cells into the lungs, abolished edema formation and inhibited hyperreactivity to substance P. Administration of Ro 45-2081 after allergen challenge reversed the influx of inflammatory cells into the lungs. Sephadex-induced neutrophil influx into the lungs of rats was also blocked by Ro 45-2081. The effects of Ro 45-2081 suggest that inhibitors of TNF may have potential as therapeutics for inflammatory diseases in the lung.

IT Allergy **inhibitors**
Anti-**inflammatory** agents
Neutrophil
Respiratory tract
(Ro 45-2081, **TNF** receptor **fusion protein**, **prevents inflammatory** responses in airways)

IT Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Ro 45-2081, **TNF** receptor **fusion protein**, **prevents inflammatory** responses in airways)

IT Lung, disease
(**inflammation**; Ro 45-2081, **TNF** receptor **fusion protein**, **prevents inflammatory** responses in airways)

IT 156679-34-4, Ro 45-2081
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Ro 45-2081, **TNF** receptor **fusion protein**, **prevents inflammatory** responses in airways)

IT 33507-63-0, Substance P
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Ro 45-2081, **TNF** receptor **fusion protein**, **prevents inflammatory** responses in airways)

L10 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
ACCESSION NUMBER: 1998:56145 CAPLUS
DOCUMENT NUMBER: 128:110576
TITLE: Ro 45-2081, a **TNF** receptor **fusion protein**, **prevents inflammatory** responses in the airways
AUTHOR(S): Renzetti, L. M.; Gater, P. R.
CORPORATE SOURCE: Hoffmann-LaRoche Inc., Nutley, NJ, 07110, USA
SOURCE: Inflammation Research (1997), 46(Suppl. 2), S143-S144
CODEN: INREFB; ISSN: 1023-3830
PUBLISHER: Birkhaeuser Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Ro 45-2081, a **TNF** receptor **fusion protein**, **prevents inflammatory** responses in the airways

L10 ANSWER 5 OF 12 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97369946 EMBASE
DOCUMENT NUMBER: 1997369946
TITLE: Ro 45-2081, a **TNF** receptor **fusion protein**, **prevents inflammatory** responses in the airways.
AUTHOR: Gater P.R.; Renzetti L.M.
CORPORATE SOURCE: P.R. Gater, Hoffmann-La Roche Inc., 340 Kingsland St., Nutley, NJ 07042, United States
SOURCE: Agents and Actions Supplements, (1997) 49/- (67-71).
Refs: 10
ISSN: 0379-0363 CODEN: AASUDJ
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Conference Article

FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
TI Ro 45-2081, a **TNF** receptor **fusion protein**, **prevents inflammatory** responses in the airways.
AB The **TNF** receptor **fusion protein**, Ro 45-2081, **inhibited** allergic and non-allergic **inflammatory** responses in the airways. Treatment of sensitized guinea-pigs with Ro 45-2081 reduced allergen-induced influx of inflammatory cells into the lungs,. . .

L10 ANSWER 6 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:137616 BIOSIS
DOCUMENT NUMBER: PREV199799436819
TITLE: **Soluble TNF receptor prevents inflammatory** disease in HCP-deficient motheaten mice with Fas-mediated apoptosis defect.
AUTHOR(S): Su, X. (1); Zhou, T.; Yang, P.; Wang, Z.; Edwardsi, C. K. Ii; Mountz, J. D.
CORPORATE SOURCE: (1) Univ. Alabama Birmingham, Birmingham, AL USA
SOURCE: Journal of Investigative Medicine, (1997) Vol. 45, No. 1, pp. 48A.
Meeting Info.: American Federation for Medical Research Southern Regional Meeting New Orleans, Louisiana, USA February 5-7, 1997
ISSN: 1081-5589.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English
TI **Soluble TNF receptor prevents inflammatory** disease in HCP-deficient motheaten mice with Fas-mediated apoptosis defect.

L10 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:258714 CAPLUS
DOCUMENT NUMBER: 124:314453
TITLE: TNF.alpha. neutralization by biological antagonists
AUTHOR(S): Bodmer, Mark W.; Foulkes, Roland
CORPORATE SOURCE: Celltech Therapeutics Ltd., Slough, UK
SOURCE: Ther. Modulation Cytokines (1996), 221-36. Editor(s): Henderson, Brian; Bodmer, Mark W. CRC: Boca Raton, Fla.
CODEN: 62QXAZ
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
IT Intestine, disease
(**inflammatory**, **TNF**.alpha. **neutralization** by **monoclonal antibodies** as therapy in **TNF** -mediated pathologies)

L10 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:669381 CAPLUS
DOCUMENT NUMBER: 123:141506
TITLE: Role of TNF.alpha. in the induction of antigen induced arthritis (AIA) in the rabbit and the anti-arthritic effect of species specific TNF.alpha. neutralizing monoclonal antibodies
AUTHOR(S): Lewthwaite, Jo; Blake, Simon; Hardingham, Timothy; Foulkes, Roland; Stephens, Sue; Chaplin, Lesley; Emtage, Spencer; Catterall, Cath; Short, Steven; et al.
CORPORATE SOURCE: Division Biochemistry, Kennedy Institute Rheumatology,

London, UK
SOURCE: Ann. Rheum. Dis. (1995), 54(5), 366-74
CODEN: ARDIAO; ISSN: 0003-4967
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Monoclonal antibodies to rabbit tumor necrosis factor .alpha. (TNF.alpha.) were developed in rats and were used to detect TNF.alpha. in synovial fluid by ELISA and to localize it in tissue sections of synovium and cartilage from rabbits up to 21 days after induction of AIA. An antibody which neutralized TNF.alpha. activity in vitro was injected into rabbits to block TNF.alpha. action in vivo in AIA. Joint swelling, leukocyte infiltration into synovium, and proteoglycan loss from cartilage were measured and compared with a control group, which were injected with sterile saline. Monoclonal antibodies to purified rabbit TNF.alpha. were prepd. in rats and 2 were selected which could neutralize rabbit TNF.alpha. in a cytotoxicity bioassay. TNF.alpha. was detected in significant concns. (21.7 pg/mL) in the arthritic joint fluid of rabbits with AIA only at one day after induction and it was then also sparsely localized in cells of the synovium, but from day 3 onwards it was localized more strongly in the deep zone of articular cartilage. Injection of anti-TNF monoclonal antibody R6 over 3 days into rabbits with AIA reduced joint swelling and leukocyte infiltration into joint fluid and decreased the expression of CD11b and CD18 on cells in the joint fluid. However, there was no redn. in the loss of proteoglycan from articular cartilage, although the joint fluid at 3 days contained a lower glycosaminoglycan content. The antibody R6 gave most effect at a dose of 0.6 mg/kg and there was no increase in its effectiveness at a 5-fold greater dose (3.0 mg/kg). Treatment over 10 days gave a more complete suppression of joint swelling, but did not result in any less proteoglycan loss from cartilage. Treatment for 5 days with a 16 day follow up gave a redn. in swelling for several days beyond the treatment, but the swelling then slowly returned, until by day 21 there was no difference in joint swelling and there was also no recovery of cartilage proteoglycan content. A rabbit anti-rat Ig response was detected at 21 days, which may have limited the long term effectiveness of the antibody. Thus, in AIA in rabbits, TNF.alpha. was only detected in synovial fluid at one day after induction and there was only limited cellular localization of TNF.alpha. in synovium and cartilage from 3 days. However, **neutralizing TNF**.alpha. with a **monoclonal antibody** was effective in suppressing **inflammatory** changes in the joint during the acute onset of AIA, but it had little effect on the loss of proteoglycan from cartilage. Apparently, blocking inflammation and synovitis with anti-TNF.alpha. may be more easily achieved than preventing damage to articular cartilage.

L10 ANSWER 9 OF 12 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96077001 MEDLINE
DOCUMENT NUMBER: 96077001 PubMed ID: 7584592
TITLE: [Increased plasma level of Type I (p55) and Type II (p75) TNF-receptors following trauma].
Erhohte Plasmaspiegel der loslichen TNF-Rezeptoren (sTNFRs) Typ I (p55) und Typ II (p75) nach Trauma.
AUTHOR: Keel M; Bonaccio M; Steckholzer U; Ungethum U; Gallati H; Trentz O; Ertel W
CORPORATE SOURCE: Departement Chirurgie, Universitatsspital, Zurich.
SOURCE: SWISS SURGERY, (1995) (5) 241-4.
Journal code: 9514313. ISSN: 1023-9332.
PUB. COUNTRY: Switzerland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124

Last Updated on STN: 19960124
Entered Medline: 19951227

AB . . . with poor outcome of injured patients. TNF-alpha seems to play a pivotal role as trigger for the induction of systemic **inflammation** . Recently, two naturally occurring **inhibitors** of **TNF** -alpha, **soluble TNF-receptors** (sTNFRs) p55 and p75, have been characterized. The present study was undertaken to determine whether severe trauma increases circulating sTNFRs. . .

L10 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:506053 CAPLUS
DOCUMENT NUMBER: 121:106053
TITLE: Anti-cytokine strategies. Modulation of systemic inflammatory response syndrome by IL-1 receptor antagonist and soluble TNF receptor
AUTHOR(S): Wakabayashi, Go; Kitajima, Masaki
CORPORATE SOURCE: Sch. Med., Keio Univ., Tokyo, 160, Japan
SOURCE: Igaku no Ayumi (1994), 169(8), 850-5
CODEN: IGAYAY; ISSN: 0039-2359
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
IT **Inflammation inhibitors**
(IL-1 receptor antagonist and **sol. TNF receptor** in sepsis in relation to)

L10 ANSWER 11 OF 12 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95121320 MEDLINE
DOCUMENT NUMBER: 95121320 PubMed ID: 7821299
TITLE: Kinetics of tumour necrosis factor-alpha, soluble tumour necrosis factor receptors, interleukin 1-beta and its receptor antagonist during serious infections.
AUTHOR: van Deuren M
CORPORATE SOURCE: Department of Internal Medicine, University Hospital Nijmegen, The Netherlands.
SOURCE: EUROPEAN JOURNAL OF CLINICAL MICROBIOLOGY AND INFECTIOUS DISEASES, (1994) 13 Suppl 1 S12-6. Ref: 41
Journal code: 8804297. ISSN: 0934-9723.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 19970203
Entered Medline: 19950216

AB . . . the central mediators in the genesis of sepsis. The proinflammatory effects of these cytokines are counteracted in vivo by natural **inhibitors**. **Soluble TNF receptors** (sTNFR) are shed upon **inflammatory** stimuli such as IL-1 beta and TNF itself. Circulating TNF can be complexed by these receptors, thus preventing TNF from. . .

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